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Foreword

The cardiomyopathies

More than 20 years ago Goodwin proposed a classification for the cardiomyopathies that for clinical and diagnostic purposes has served cardiologists well. The original definitions were based on morphology and function and were purely descriptive. Recent technological advances in molecular biology and immunology have permitted examination of the molecular genetic basis of hypertrophic cardiomyopathy (HCM) and of the viral and immunological pathogenesis of dilated cardiomyopathy (DCM). These new developments prompted a two day international workshop (12–14 May 1993) on the cardiomyopathies that was held in La Coruña, Spain, and sponsored by the International Society and Federation of Cardiology, the European Society of Cardiology, and the Galician Health Service. The impetus for the meeting came from collaborative efforts of the guest editors to find the gene responsible for HCM in a large Galician family with apical hypertrophic cardiomyopathy. The meeting targeted new developments in the understanding of the aetiology and pathogenesis of the cardiomyopathies as well as controversies and problems related to clinical management.

The identification of missense mutations in the myosin gene in patients with HCM should lead to disease-causing mutations being introduced into transgenic animals and the examination of the phenotype. There are many questions to examine: Why do most patients with HCM have left ventricular hypertrophy? Is myocyte disarray the result of sarcomere instability? Is the accepted hypothesis that myocardial disarray provides the substrate for electrical instability correct? Do the extent and distribution of myocardial disarray alter during life—in particular at puberty, when the incidence of sudden death is greatest?

Predictably, new discoveries raise more questions than they resolve. In dilated cardiomyopathy the ability to detect viral genome in myocardium and to assess tissue markers of immune activation may make endomyocardial biopsy invaluable in the assessment of disease activity and the clinical characterisation of patients. The finding of familial disease in over 25% of patients and the production of pedigrees consistent with autosomal dominant inheritance has led many centres to perform linkage analysis to identify the genes responsible. These are exciting times for students of the cardiomyopathies. These developments are merely the opening shots in the campaign to understand the aetiology and pathogenesis of hypertrophic and dilated cardiomyopathy: they signify a new era and the potential to base classification on molecular genetics and viral/immune activity.

This supplement of the *British Heart Journal* presents selected topics that reflect the new directions in the investigation of the basis of the cardiomyopathies and, where possible, their clinical application. The supplement has been jointly sponsored by the British Cardiac Society and the Sociedad Española de Cardiologia and was made possible through a grant from the Consejeria de Sanidad e Servicios Sociales de Galicia. As guest editors we are pleased to present these papers, especially because the original description of what was then called Teare's disease was first published in the *British Heart Journal.*

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